A110.E1031
JACC March 9, 2010
Volume 55, issue 10A

MYOCARDIAL ISCHEMIA AND INFARCTION

CARDIOPROTECTIVE EFFECTS OF α1-ANTITRYPSIN IN EXPERIMENTAL ACUTE MYOCARDIAL INFARCTION DUE TO TRANSIENT ISCHEMIA IN THE MOUSE

ACC Poster Contributions
Georgia World Congress Center, Hall B5
Sunday, March 14, 2010, 3:30 p.m.-4:30 p.m.

Session Title: Myocardial Energetics and Protection
Abstract Category: Myocardial Ischemia/Infarction--Basic
Presentation Number: 1104-304

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Background: α1-Antitrypsin (AAT) is the major serum serine-protease inhibitor. In the current study we tested the effects of exogenous human AAT on left ventricular remodeling and function after acute myocardial infarction (AMI) in the mouse.

Methods: Adult male ICR mice were randomly assigned to treatment with AAT (2 mg, i.p) or saline in AMI induced by surgical coronary artery ligation for 30 minutes and reperfusion (N=6 per group). Transthoracic echocardiography was performed at baseline and 7 days after surgery for measuring of LV end-diastolic (LVEDD) and systolic (LVESD) diameters, and LV fractional shortening (LVFS). Infarct scar size was measured by pathological examination. Plasma levels of the monocyte chemoattractant protein-1 (MCP-1) were determined in additional groups of AAT- or saline-treated mice 6 hours after AMI (N=4 per group).

Results: After AMI, mice treated with AAT exhibited a 90% smaller increase in LVEDD, 55% smaller increase in LVESD, and 55% smaller decrease in LVFS ($p<0.05$ vs saline for all comparisons). There was a 47% smaller infarct scar in the LV in AAT-treated mice ($p<0.05$ vs Saline). MCP-1 levels were also significantly reduced in AAT-treated mice (79±30 vs 2±2 pg/ml, $p<0.05$ vs saline).

Conclusions: Exogenous administration of AAT during AMI leads to preservation of viable myocardium and prevention of adverse cardiac remodeling